nous epinephrine, diphenhydramine, aminophylline, and methylprednisolone sodium succinate for presumed anaphylaxis to the liniment used in the acupuncture. A chest x-ray study was done that showed bilateral, nearly complete pneumothoraces. Bilateral chest tubes were placed, which resulted in a prompt resolution of the hypotension and respiratory distress. He was discharged well several days later and was thought to have no underlying pulmonary disease.

This case, and that reported by Dr Wright and associates, indicate a potentially lethal complication of thoracic acupuncture. I am also aware of a number of patients who have had unilateral pneumothorax related to attempted corticosteroid injection of the shoulder or thoracic trigger points. Practitioners in any field of medicine who use thoracic injection techniques must exercise caution in the depth of insertion and be aware of the potential for pneumothorax.

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REFERENCE

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Carbon Monoxide Poisoning

To the Editor: We read with interest the report by Gasman and colleagues regarding an indoor barbecue and carbon monoxide poisoning in the December 1990 issue. We have encountered a similar group of patients that underscores some of the authors' points:

Two 31-year-old non-English-speaking women were brought to the emergency department by paramedics, who suspected the patients had food poisoning. They had abdominal pain, nausea, vomiting, dizziness, and headache. One had had a single episode of diarrhea. They had shared a meal of barbecued beef. Three other members of the household had experienced similar but milder symptoms that did not require treatment.

The results of a physical examination of both patients were normal. On further questioning, the women admitted that the beef had been prepared on a barbecue stove in their closed apartment. They had been reluctant to provide this information to the paramedics because, having been in the United States for only a brief period of time, they assumed the paramedics—because of their uniforms—had police functions.

Once this item of history had been obtained, the patients were placed on 100% oxygen therapy. Carboxyhemoglobin levels were measured and found to be 38.3% and 31.9%. The other members of the household were called and evaluated. All had normal findings on physical examinations. Carboxyhemoglobin levels were 25.5%, 21.2%, and 18.5%.

These cases underscore the similarity that carbon monoxide poisoning may display to food poisoning, especially when more than one patient is involved. The difficulty of obtaining an accurate history because of language barriers and social background may enhance the challenge of arriving at the correct diagnosis.

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REFERENCE

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TO THE EDITOR: We read with great interest the report by Gasman and co-workers regarding carbon monoxide (CO) poisoning in a family using charcoal for indoor cooking purposes.¹ The article did an excellent job of highlighting a recurrent and insidious environmental health hazard. The authors made a few technical points, however, that merit further comment.

The authors quote a normal range for carboxyhemoglobin (COHb) saturation of less than $0.150 \,(<15\%)$. Most observers agree that COHb levels as low as 10% to 15% can be responsible for headaches and dizziness. A 1989 study implicated carboxyhemoglobin levels as low as 2% in reducing exercise tolerance among patients with coronary artery disease and angina. Further, in a large population-based survey, 95% of nonsmokers were found to have COHb saturations below $0.02 \,(<2.0\%)$ and 95% of smokers below $0.085 \,(<8.5\%)$. Hence, more reasonable "normal" ranges are 0.02 or less ($\leq 2.0\%$) for nonsmokers and below $0.09 \,(<9.0\%)$ for smokers, with an irreducible minimum of 0.003 to $0.005 \,(0.3\%$ to 0.5%) due to porphyrin catabolism.

The authors also state that dissolved CO combining with cytochromes, not impaired oxygen delivery due to COHb formation, is responsible for the toxic effects of CO. They base this assertion on a 1976 report in which anemic dogs transfused with CO-saturated blood failed to show signs of CO toxicity. In studies of experimental animals perfused with a hemoglobin substitute (a fluorinated compound without special affinity for CO), however, the animals tolerated atmospheres of 3% to 5% CO—environments that would have been rapidly fatal had the primary mechanism of CO toxicity been the interaction of dissolved CO with cytochromes.⁴

The authors make the point that "patients with severe neurologic or cardiovascular symptoms or very high COHb concentrations would benefit from hyperbaric oxygen." Although hyperbaric oxygen does substantially hasten the elimination of CO and the reduction of COHb levels in CO poisoning, there have yet to be any controlled studies showing that the ultimate outcome in patients treated with hyperbaric oxygen is better than in patients treated with normobaric oxygen.

Concern over the type of event outlined in Dr Gasman's report prompted the California Department of Health Services to issue a public warning in January 1990 cautioning against the use of gas ranges and unvented gas or kerosene heaters for indoor heating, as well as the practice of using charcoal for indoor cooking. The advisory especially targeted Asian immigrants because the traditional use of charcoal for indoor cooking purposes has been previously reported within this community. We are appreciative of Dr Gasman and colleagues for again bringing this issue to the forefront.

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